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Hepatectomy and Arterial Blood Ketone-Body Ratio II. Clinical Significance of Arterial Blood Ketone-Body Ratio in Hepatectomized Patients

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Abstract

Twenty-five hepatectomized patients, both cirrhotic and noncirrhotic, were studied for the postoperative changes in the arterial blood ketone-body ratio (acetoacetate/3-hydroxybutyrate) and the standard liver function tests. It was found that the blood ketone-body ratio was more closely correlated to the mortality and morbidity rates than the conventional liver function tests. Therefore, it is proposed that this parameter enables more effective postoperative management by the accurate evaluation of hepatic functional reserve after hepatectomy.

Introduction

To provide effective postoperative management after hepatectomy, it is of great importance to determine accurately the critical period of impaired hepatic function[9]. To this end, the standard liver function tests are routinely performed on hepatectomized patients. In recent years, however, more accurate means of evaluating the decreased functional reserve of the remnant liver have been sought to complement the standard liver function tests. Experimental studies on the metabolic aberrations occurring in the remnant liver have indicated that the hepatic functional reserve is closely correlated with the hepatic energy charge level (ATP+1/2 ADP)/(ATP+ADP+AMP)[4,18]. More recent studies using hepatectomized rabbits have shown that the hepatic energy charge correlates positively with the ketone-body ratio (acetoacetate/3-hydroxybutyrate) of arterial blood[3].

In the present study involving cirrhotic and noncirrhotic patients, the changes in the ketone-body ratio of arterial blood following hepatectomy were examined in relation to major postoperative complications. Evidence will be presented indicating that, compared with conventional liver function tests, the arterial blood ketone-body ratio could serve as a relatively more reliable parameter for the clinical evaluation of metabolic dysfunction after hepatectomy.

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Key words: Hepatectomy, Postoperative complications, Blood ketone-body ratio, Liver function tests.
索引語：肝切除，術後合併症，血中ケトン体比，肝機能検査.
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Materials and methods

Twenty-five patients with resectable liver tumor (table 1) were admitted to the 1st Department of Surgery of Kyoto University Hospital during the 2.5-year period beginning in March 1979. There were 14 males and 11 females whose ages ranged from 47 to 82 years with a mean age of 58 years, with the exception of a 10-year-old girl. The liver tumors were classified histologically into 19 hepatomas, 4 metastatic liver tumors from colonic or gastric cancer, 1 cholangioma and 1 benign tumor (hemagioma). Liver cirrhosis with hepatoma was found in 12 patients who underwent 5 major (lobular or more) and 7 minor (sublobular) resections. The 13 noncirrhotic patients had 12 major and 1 minor resections. In addition, 42 subjects consisted of 7 patients without liver disease and 35 patients with liver disease such as liver cirrhosis with hepatoma and/or esophageal varices, and obstructive jaundice were used for the preliminary

Table I. Clinical Material

Patient number	Age Sex	Primary disease	Coexistent cirrhosis	Type of resection
Group A				
1	10 F	hepatoma[1]	no	extensive right lobectomy
2	48 M	hepatoma	no	right lobectomy
3	52 F	hepatoma	yes	partial resectin of right lobe[2]
4	53 M	hepatoma	yes	left lateral segmentectomy
5	57 M	hepatoma	no	extensive right lobectomy
6	57 F	cholangioma	no	partial resection of right lobe[2]
7	59 M	hepatoma	no	left lobectomy
8	63 M	hepatoma	yes	partial resection of right lobe[2]
9	63 F	metastatic liver tumor	no	left lobectomy
10	74 M	metastatic liver tumor	no	right lobectomy
Group B				
11	50 F	hepatoma	yes	partial resection of right lobe[2]
12	50 F	metastatic liver tumor	no	extensive right lobectomy and partial colectomy
13	51 M	hepatoma	no	left lobectomy
14	54 M	hepatoma and gastric cancer	no	right lobectomy and partial gastrectomy
15	56 F	hepatoma	yes	right lobectomy
16	64 F	hepatoma	no	right lobectomy
17	65 F	hepatoma	yes	extensive right lobectomy
18	65 M	benign liver tumor	no	right lobectomy
19	82 M	metastatic liver tumor	no	right lobectomy and right hemicolectomy
Group C				
20	47 F	hepatoma	yes	left lateral segmentectomy
21	50 M	hepatoma	yes	right lobectomy
22	52 F	hepatoma	yes	right lobectomy
23	54 M	hepatoma	yes	right lobectomy
24	60 M	hepatoma	yes	partial resection of right lobe[2]
25	65 M	hepatoma	yes	partial resection of right lobe[2]

[1] The histopathology of the tumor of this pediatric case was identical to adult hepatocellular carcinoma.
[2] Includes wedge resection and enucleation.

study of the postoperative changes in blood ketone-body ratio. The ages of 7 patients without liver disease (4 men and 3 women) ranged from 39 to 79 years with a mean age of 53 years, while the ages of 35 patients with liver disease (19 men and 16 women) ranged from 35 to 75 years with a mean age of 49 years. No patients were diabetic as to require treatment with insulin or any other agent.

After informed consent from these patients, 4 ml of arterial blood were obtained postoperatively with a heparinized syringe via the femoral artery. For reasons mentioned later, blood samples were taken directly at 1 hr after a 2-hour intravenous infusion with 10% glucose in distilled water to maintain their blood glucose levels at 120–200 mg/dl. The rate of glucose infusion was 0.5 g per kilogram body weight per hour. All other medications were discontinued for 4 hr prior to the removal of arterial blood during the glucose infusion. For the assay of ketone bodies, 3 ml of arterial blood were immediately mixed with the same volume of ice-cold 10% (wt/vol) perchloric acid. The suspension was centrifuged at 10,000×g for 15 min at 0–4°C. The supernatant was adjusted to a pH of 7.0 with 69% (wt/vol) K_2CO_3 and recentrifuged at 10,000×g for 5 min at 0–4°C. The final supernatant was used to determine the concentrations of ketone bodies, acetoacetate and 3-hydroxybutyrate, which were measured spectrophotometrically by a modification of the standard methods of Mellanby and Williamson[7] and Williamson and Mellanby[17], respectively. The remaining arterial blood was used for blood gas analysis, because hypoxia is an important factor which directly affects the blood ketone-body ratio[13]. However, a hypoxic state (less than 60 mmHg of pO_2) was not detected in any patient at sampling time.

Venous blood analyses of serum enzyme activities, total protein, albumin, globulin, total bilirubin, total cholesterol and prothrombin time were carried out pre- and postoperatively, according to the conventional methods at the Central Laboratory of Kyoto University Hospital. The enzymes investigated in this study were glutamic oxaloacetic transaminase, glutamic pyruvic transaminase, lactic dehydrogenase, and alkaline phosphatase.

Results

Figure 1 summarizes the preliminary study of the postoperative determinations of blood ketone bodies in 7 patients without liver disease and 35 patients with liver disease. The blood ketone-body ratios of 7 patients without liver disease were above 0.7, while those of 7 patients with liver disease and operative death were below 0.7, especially below 0.4. The blood ketone-body ratios of 28 patients with liver disease who survived operation were not below 0.4. In addition, this overall plotting shows that, although not so clearly, the blood ketone-body ratios were inversely proportional to the amounts of total ketone bodies.

As shown in table 1 and figure 2, the hepatectomized patients were classified into three groups (A–C) according to the postoperative changes in the blood ketone-body ratio. In Group A (patients 1–10), major resections were performed on 6 noncirrhotic patients, and 4 minor ones on 3 cirrhotic and 1 noncirrhotic patients. The blood ketone-body ratios of this group were above 0.7 during the postoperative period. In Group B (patients 11–19), 8 major resections were

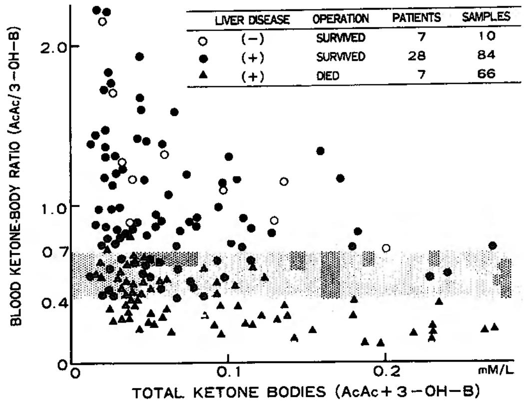

Fig. 1. Postoperative determinations of arterial blood ketone-body ratios (vertical bar) and total ketone bodies (horizontal bar) in 42 subjects consisted of 7 patients without liver disease and 35 patients with liver disease.

performed on 6 noncirrhotic and 2 cirrhotic patients, with the exception of 1 minor resection in 1 cirrhotic patient. The blood ketone-body ratios of this group remained between 0.4 and 0.7 in the postoperative period, and then rose to above 0.7 within the first 2–10 days. Group C patients (patients 20–25), all of whom were cirrhotic, underwent 3 major and 3 minor resections. The blood ketone-body ratios of this group showed a prolonged decrease to below 0.7 and eventually, a marked decrease to below 0.2 without restoration to above 0.7.

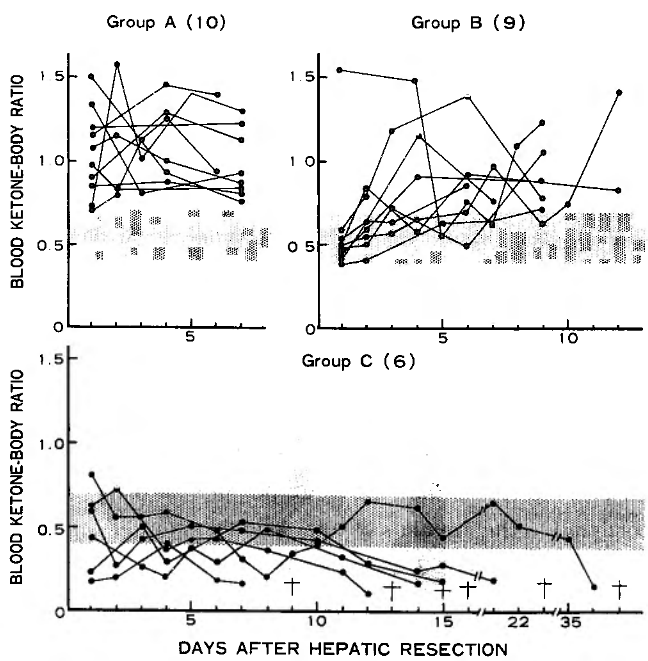

Fig. 2. 25 hepatectomized patients were divided into three groups according to the postoperative changes in the arterial blood ketone-body ratio. The shadowed portion indicates the range between 0.4 and 0.7.

Table II. Major Postoperative Complications in Each Group

Complications	Group A (10)	Group B (9)	Group C (6)
Pulmonary complications	0	4	6
Intraperitoneal bleeding	0	1	3
Upper GI bleeding	1	0	5
Severe jaundice[1]	2	4	6
Coagulation defects[2]	0	1	6
Renal insufficiency[3]	0	1	6
Mental disturbance	0	1	6
Operative death	0	0	6

[1] Severe jaundice was defined as serum bilirubin level>7 mg%.
[2] Coagulation defects were defined as platelet counts <50,000/ml, blood fibrinogen level<100 mg% or prothrombin time>25 s.
[3] Renal insufficiency was defined as blood urea nitrogen>50 mg% or serum creatinine>3mg%. Numbers of patients in each group are shown in parentheses.

The major postoperative complications of each group are summarized in Table 2. There were few complications in group A. The massive hemorrhage from a gastric ulcer, observed in patient 8, was successfully treated by replacement transfusion and antacid therapy. In group B, a variety of postoperative complications occurred, but most of them were ameliorated concomitantly with the restoration of the blood ketone-body ratio, as described later. In group C, however, the repeated occurrence of postoperative complications was observed along with prolonged and irreversible decreases in the blood ketone-body ratio. Finally, all patients of this group died of multiple organ failure.

The postoperative changes in serum enzyme activities of each group are shown in figure 3. In all the groups, glutamic oxaloacetic transaminase, glutamic pyruvic transaminase and lactic dehydrogenase showed sharp elevations on the first postoperative day, and thereafter declined rapidly to near the preoperative levels within the 1st week. In contrast, the preoperative increase in alkaline phosphatase of group A and C decreased postoperatively, while in group B it remained within the normal range pre- and postoperatively.

The postoperative changes in serum total protein, albumin, total bilirubin and total cholesterol of each group are shown in figure 4. Total protein fell rapidly 24 hr after the operation in all groups, and it returned to normal on the 2nd day in group B, the 5th to 7th days in group A, and the 8th to 10th days in group C. The rapid normalization in group B was due to the administration of large quantities of albumin solutions, as corroborated by the serum albumin level of this group which was only one which did not decrease postoperatively. Total bilirubin increased in all groups 2 days after the operation, and thereafter decreased gradually in group A and B, while a progressive increase was observed in group C. Total cholesterol decreased rapidly after the operation and remained uniformly reduced after the 1st week in all three groups.

Brief summaries of the clinical course of typical group B and C patients are as follows. Figure 5 shows the postoperative course of group B patient 15, a cirrhotic patient who underwent

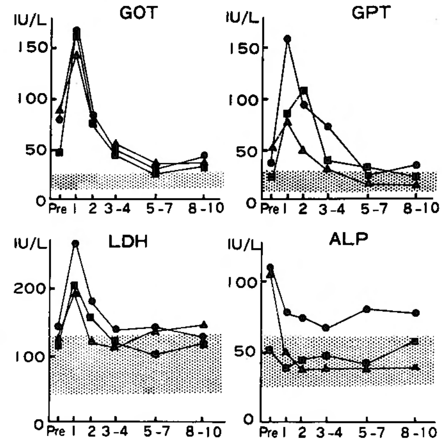

Fig. 3. Postoperative changes in serum enzyme activities of three groups excluding the recent cases (patients 2, 9, 12, 17 and 25). Each point represents the mean value. The shadowed portion indicates the normal range of individual enzyme. GOT=glutamic oxaloacetic transaminase; GPT=glutamic pyruvic transaminase; LDH=lactic dehydrogenase; ALP=alkaline phosphatase.
●=group A; ■=group B; ▲=group C.

a right lobectomy for a large hepatoma. The arterial blood ketone-body ratio decreased rapidly to 0.5 on the 1st postoperative day and remained between 0.5 and 0.7 until the 6th day. Adult respiratory distress syndrome developed on the 3rd day, which was successfully treated by positive end-expiratory pressure breathing over 4 days. During this period, serum bilirubin remained below 6 mg% and prothrombin time below 16 sec. Serum albumin level was main-

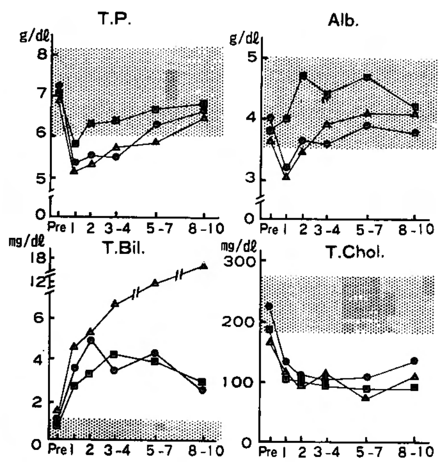

Fig. 4. Postoperative changes in serum total protein, albumin, total bilirubin and total cholesterol of three groups excluding the recent cases (patients 2, 9, 12, 17 and 25). Each points represents the mean value. The shadowed portion indicates the normal range of individual parameters.
●=group A; ■=group B; ▲=group C.

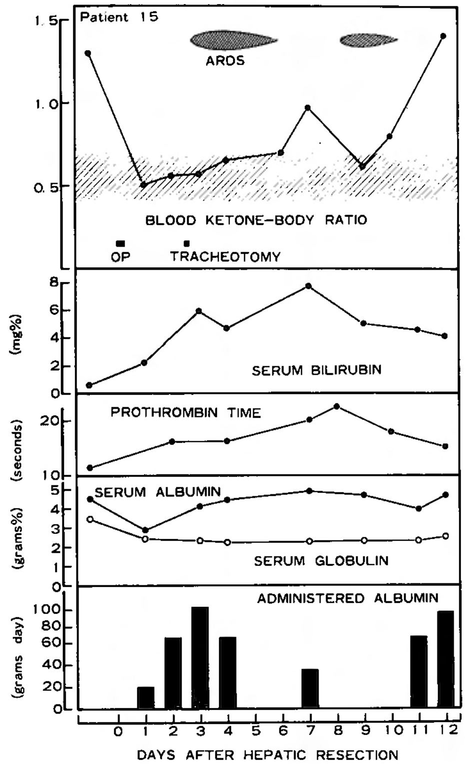

Fig. 5. Postoperative clinical course and laboratory data of group B patient 15. OP: right lobectomy. ARDS=adult respiratory distress syndrome; DIC=disseminated intravascular coagulation.

tained above 4 g% by the supplemental albumin administrations, while serum globulin level remained reduced. On the 7th day, the blood ketone-body ratio increased transiently to above 0.7. However, the occurrence of disseminated intravascular coagulation set in on the 8th day, with blood analyses showing 47,000/ml platelets, a fibrinogen level of 93 mg% and more than 40 μg/ml of fibrinogen degradation products. The blood ketone-body ratio decreased again to below 0.7. General heparinization was promptly performed for 3 days. On the 12th day, fibrinogen degradation products decreased to normal concomitantly with the sharp rise of blood ketone-body ratio. Thereafter, the patient had an uneventful recuperative course, although mild jaundice, increased prothrombin time, prolonged hypoglobulinemia and hypoalbuminemia treated by albumin administration were still observed on the 12th day.

Figure 6 demonstrates the clinical course of group C patient 20, who had no obvious complications during 13 days after a left lateral segmentectomy on a severely cirrhotic liver. During this period, serum bilirubin was below 3.5 mg% and prothrombin time was not increased to more than 20 sec. Hypoalbuminemia was effectively treated by albumin administration, while serum globulin level remained markedly reduced. It should be noted that the arterial blood ketone-body did not rise.

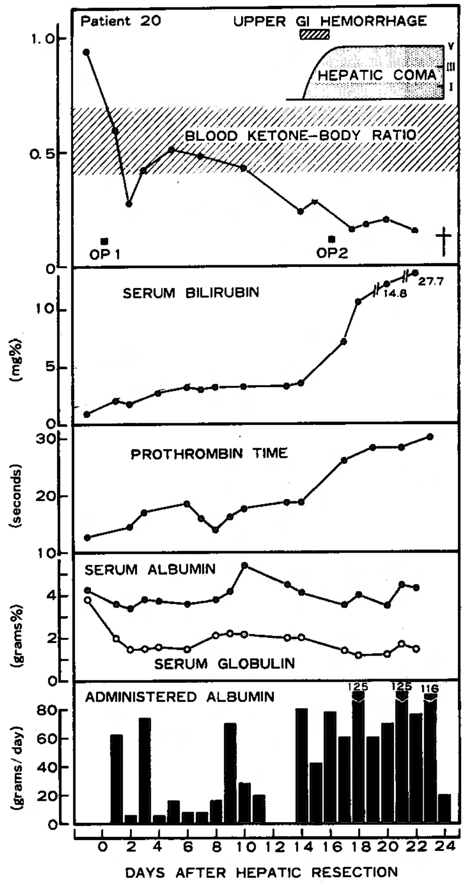

Fig. 6. Postoperative clinical course and laboratory data of group C patient 20. OP 1= left lateral segmentectomy; OP 2=total gastrectomy.

Discussion

Following hepatectomy, considerable changes occurred in serum enzyme activities, total protein, albumin, globulin, total bilirubin, total cholesterol and prothrombin time, which were consistent with the previous reports[1,2,6]. However, these parameters, while useful as the indicators of metabolic changes in the hepatectomized patients, did not give a direct and accurate evaluation of the decreased functional reserve of the remnant liver underlying the onset of hepatic insufficiency. By contrast, the blood ketone-body ratio was closely correlated to the mortality and morbidity rates in the hepatectomized patients. In group B patients with a transient decrease to 0.4–0.7 of the blood ketone-body ratio, major postoperative complications were ameliorated with the restoration of blood ketone-body ratio. However, all group C patients with blood ketone-body ratios below 0.4 died of hepatic insufficiency without restoration of blood ketone-body ratio.

The basic biochemical mechanism behind the blood ketone-body ratio may be depicted as follows. The acetoacetate to 3-hydroxybutyrate ratio in the tissue reflects the oxidoreduction state of free nicotinamide adenine dinucleotides in mitochondria, as shown in the following formula[16]: acetoacetate+NADH+$H^+ \rightleftarrows$ 3-hydroxybutyrate+NAD. Thus, free NAD/free NADH=1/K$\times$acetoacetate/3-hydroxybutyrate, where K is the equilibrium constant of 3-hydroxybutyrate dehydrogenase, the activity of which is exceptionally high in liver mitochondria. It is well known that these ketone bodies pass readily through the cell membrane into the blood stream and reach the peripheral tissues where they are oxidized via the tricarboxylic acid cycle. Therefore, the ketone-body ratio in arterial blood can be considered to reflect the oxido-reduction state of free nicotinamide adenine dinucleotides in liver mitochondria, which is closely linked to various steps in the mitochondrial energy-generating systems. The blood ketone-body ratio depends on the oxidation of glucose or fatty acids in liver mitochondria[10,11]. When a normal subject takes sufficient glucose, the blood ketone-body ratio is high, with an increase in NAD, associated with glucose oxidation in liver mitochondria. Under fasting, however, the mobilized fatty acids are oxidized in liver mitochondria, producing excess NADH in mitochondria with a resultant decreased blood ketone-body ratio. However, the decreased blood ketone-body ratio under fasting is rapidly restored after the administrtion of sufficient glucose. By contrast, a recent study from our laboratory has shown that the decreased blood ketone-body ratio in hepatectomized rabbits was not restored by glucose administration[14]. Hence, in the present study, to exclude the fasting effect, the blood ketone-body ratio of hepatectomized patients was assayed under sufficient glucose supply.

With regard to the decrease of the blood ketone-body ratio in the hepatectomized patients, there are two possible mechanisms. The first is an enhancement of the β-oxidation of fatty acids associated with the production of excess NADH. The second is an inhibition of the electron transport system due to mitochondrial impairment. In hepatectomized rabbits, the blood ketone-body ratio decreased along with the increasing degrees of resection[3]. In 25% hepatectomy, the blood ketone-body ratio decreased only slightly, while, in 70% hepatectomy, the blood ketone-body ratio decreased rapidly to 0.4 before returning to a normal level, as observed in group B patients. Mitochondrial phosphorylative activity of the remnant liver in 70% hepatectomy was enhanced remarkably in order to restore the markedly decreased hepatic energy charge level by providing ATP in quantities. At this stage, the liver mitochondria preferentially oxidized fatty acids as an efficient energy source, rather than glucose, resulting in a drop in the blood ketone-body ratio[8]. In 93% hepatectomized rabbits, analogous to group C patients, the blood ketone-body ratio decreased drastically concomitantly with the advent of mitochondrial impairment in the phase immediately after the operations, resulting in total mortality as the blood ketone-body ratio approached 0.2. These experimental observations indicate that, with increasing metabolic loads upon the remnant liver, the blood ketone-body ratio is first decreased moderately by the enhancement of fatty acid oxidation and then markedly by mitochondrial impairment. In addition, the blood ketone-body ratio of 0.4 seems to be the critical point at which irreversible metabolic alterations occur in the remnant liver after hepatectomy. This appears to be consistent with the critical value observed between groups B and C. From these considerations, the transient decrease in the blood ketone-body ratio in group B may indicated that an enhancement of fatty acid oxidation in liver mitochondria is taking place to overcome the decreased energy charge level of the remnant liver. By contrast, the irreversible decrease in the blood ketone-body ratio in group C suggests an inhibition of the electron transport system due to the serious mitochondrial impairment, resulting in an irreversibly decreased hepatic energy charge. All group C patients succumbed when the blood ketone-body ratio decreased progressively to approximately 0.2.

On the other hand, it has been found that decreases in hepatic energy charge were positively correlated with decreases in blood ketone-body ratios in hepatectomized animals[3]. Therefore, the decrease of blood ketone-body ratio discussed in this study may be characterized in terms of the progressive decrease of hepatic energy charge levels. The mechanism may be explained by the following hypothesis[5,12]: the decreasing blood ketone-body ratios are consistent with a progressive reduction in mitochondrial oxido-reduction state. The reduced mitochondrial oxido-reduction state inhibits the citrate system, which decides the turnover rate of the tricarboxylic acid cycle, and the processes requiring NAD^+ in the mitochondria such as pyruvate dehydrogenase, isocitrate dehydrogenase and α-ketoglutarate dehydrogenase[15]. The overall picture of the decrease in blood ketone-body ratio is consistent with a progressive inhibition of the turnover rate of the tricarboxylic acid cycle, resulting in a marked hepatic energy deficit. By documenting the various metabolic states existing up to the terminal one, it is likely that the arterial blood ketone-body ratio will gain wide clinical application as a simple and accurate means of determining the immediate state of metabolic dysfunction in the remnant liver, upon which effective postoperative management depends.

Summary

Twenty-five hepatectomized patients, both cirrhotic and noncirrhotic, were studied for the postoperative changes in the arterial blood ketone-body ratio and standard liver function tests. It was found that serum enzyme activities, total protein, albumin, total bilirubin, total cholesterol and prothrombin time were less reliable direct indicators of the onset of postoperative hepatic insufficiency than the arterial blood ketone-body ratio, which reflects the oxido-reduction state of liver mitochondria. The hepatectomized patients were classified into three groups according to the postoperative changes in the blood ketone-body ratio. In the 10 patients of group A, the blood ketone-body ratio did not decrease to below 0.7. In the 9 patients of group B, the blood ketone-body ratio decreased transiently to 0.4–0.7 and then increased to over 0.7. In the 6 patients of group C, the blood ketone-body ratio decreased irreversibly to below 0.4. There were few postoperative complications in group A. In group B, while a variety of complications occurred, they were ameliorated concomitantly with the restoration of the blood ketone-bcdy ratio. All group C patients died of multiple organ failure. It is proposed that the arterial blood ketone-body ratio is a new and practical approach which enables more effective postoperative management by the accurate evaluation of hepatic functional reserve after hepatectomy.

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和文抄録

肝切除と動脈血中ケトン体比
II. 肝切除後の動脈血中ケトン体比の臨床的意義

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動物実験（上記第I編）において，肝切除後の残存肝機能はそのエネルギーチャージに依存し，これは動脈血中ケトン体比の変動に鋭敏に反映されることが明らかになった．そこで本研究では，肝切除症例の術後血中ケトン体比の変動を測定し，一般肝機能検査と比較検討した。測定しえた肝切除症例25例は，血中ケトン体比の術後変動によって3群に分類された．すなわち，血中ケトン体比が0.7以上の高値で経過したA群（10例），術後一過性に0.4-0.7へ低下したB群（9例），および0.4以下へ不可逆的に低下したC群（6例）である．A群では合併症が少なく順調な術後経過をたどったが，B群では血中ケトン体比の低下時に合併症が多発し，0.7以上へ上昇するとともにこれら合併症の軽快をみた．一方，C群では重篤な合併症が頻発し，全例多臓器不全の病像を呈し死亡した．また，これら血中ケトン体比の変動は一般肝機能検査と比較して，臨床経過をより鋭敏に反映していることが明らかになった．